adjacent to the important small joints of the hands, might well prevent retention of calcite or epidermal protein and thus avoid the serious sequelae of chronic inflammatory reaction. The use of antibiotics for control of bacterial infection should be gauged on the same general principles applicable to other types of wounds.

The problem of fusiform swelling of a finger, particularly about the middle or proximal interphalangeal joint, is a common one. Usually examination will show limited joint motion, and laxity or crepitation within the joint may be noted. There is usually some local redness and heat which goes along with the swelling. Early x-ray films are often not revealing but later will show joint narrowing and areas of decalcification on either side. In the presence of pain or instability, splinting of the affected joint generally gives prompt relief of symptoms and a chance for the lesion to abate if it is of a self-limiting nature.

Failure of favorable response under a reasonable trial of conservative therapy is an indication for surgical exploration and biopsy of the joint synovium or of bone if involved. A specific diagnosis may be made thereby, and specific treatment rendered. If the disease leaves the joint so disabled or painful as to be nonfunctioning, then surgical arthrodesis in approximately 45° of flexion can generally restore reasonably good use of the digit.

Summary

A case is described of a SCUBA diver with a chronic, disabling inflammatory reaction in his finger at the site of a puncture wound from a sea urchin spine. It is concluded that early treatment should consist of open surgical exploration and removal of spine fragments, to prevent, if possible, the development of a chronic granulomatous reaction and permanent disability.

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REFERENCES

1. Earle, K. V.: Echinoderm injuries in Nauru, Med. J. Australia; 2(10):265-266, 1941.

2. Endean, R.: The venomous sea urchin toxopneustres pileolus, Med. J. Australia, p. 320, March 4, 1961.

3. Rocha, G., and Fraga, S.: Sea urchin granuloma of skin, Arch. Derm., 85:406, 1962.

Tumor of the Duodenum Removed in the Course of Partial Gastrectomy For Duodenal Ulcer

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ZOLLINGER AND ELLISON were the first to relate the non-insulin producing islet-cell tumor of the pancreas to a peptic ulcer diathesis. These ulcers are often atypically located, are associated with large volumes of highly acid gastric juice and are refractory to the usual method of therapy. Since the publication of the original paper in 1955,[8] there has been considerable interest in the syndrome and additional cases have been added to the world literature. More than a hundred have been reported. Oberhelman and coworkers[5] in 1961 described six cases of ulcerogenic tumors of the duodenum which were considered to be misplaced, non-insulin secreting islet-cell tissue identical to the adenomas occuring within the confines of the pancreas, and they found that removal of the adenoma in the wall of the duodenum resulted in dramatic relief of the strong ulcerogenic tendency. Since his original report, Oberhelman has observed three additional cases and apparently Ellison has recently had his first.[4] Since the condition is so rarely encountered, a report of another patient with adenoma of the duodenum associated with duodenal ulcer seems indicated.

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Report of a Case

A 48-year-old Negro woman first started to have abdominal pain at the age of eight and by the time she was 13 she was having obstructive symptoms as well as pain. A roentgenological study showed a duodenal ulcer. Considerable improvement in symptoms was brought about by conservative treatment and she had no further major trouble until at age

16 she had an episode of fainting secondary to hemorrhage from the ulcer and was put in hospital for six weeks. Roentgenographic studies again showed evidence of a duodenal ulcer at the age of 28. Conservative therapy was carried out and healing was later demonstrated. "Ulcer pain" recurred from time to time, the patient taking antacid agents for relief. At the age of 33 she was admitted to a hospital with another episode of upper gastrointestinal bleeding and radiological studies showed pronounced deformity of duodenal bulb and a definite ulcer crater. Six weeks later the crater was no longer visible. During a month in hospital the patient received one blood transfusion as well as conservative treatment for ulcer. No gastric analysis was carried out at that time nor, as far as could be determined, at any previous time. Conservative management became less and less effective, the patient having a number of periods of symptoms of obstruction, and pyloric obstruction was demonstrated by roentgenogram. She was put in hospital several times because of this complication. Finally she had constant epigastric pain, only partly relieved by food and therapeutic agents. After a year of refractory symptoms, she elected surgical intervention. She gave no history of diarrhea at any time.

She was operated on in 1954 at the age of 39. Considerable scarring and narrowing was noted at the pylorus and in the first portion of the duodenum. There was sufficient normal duodenum beyond the scarred and ulcerated area to permit gastroduodenostomy, however, and the surgeon estimated that he removed three-fifths of the stomach. No note was made as to the condition of the pancreas; the liver was considered to be normal. The resected portion of the stomach measured 15 cm along the greater curvature. Microscopic examination of the distal portion of the specimen revealed gastritis of the antrum of the stomach, and the duodenum contained a chronic ulcer surrounded by considerable fibrosis. In an adjoining area, beneath a cluster of Brunner's glands, there was a well differentiated epithelial structure composed of interlacing double cords of cells with uniform oval nuclei and rather scanty cytoplasm devoid of granules. There were clusters of lymphocytes scattered through this lesion and it was considered at that time to be a hamartoma of the duodenal wall, probably of pancreatic origin. When the case was reviewed in detail in 1963, nine years later, the report of the duodenal hamartoma was noted and review of the slides revealed the lesion to be a pancreatic adenoma located in the duodenal wall (Figures 1, 2 and 3) and morphologically consistent with the ulcerogenic pancreatic adenoma as described by Zollinger and Ellison and others.

After the operation she had little "ulcer pain," and what pain she had responded readily to anti-

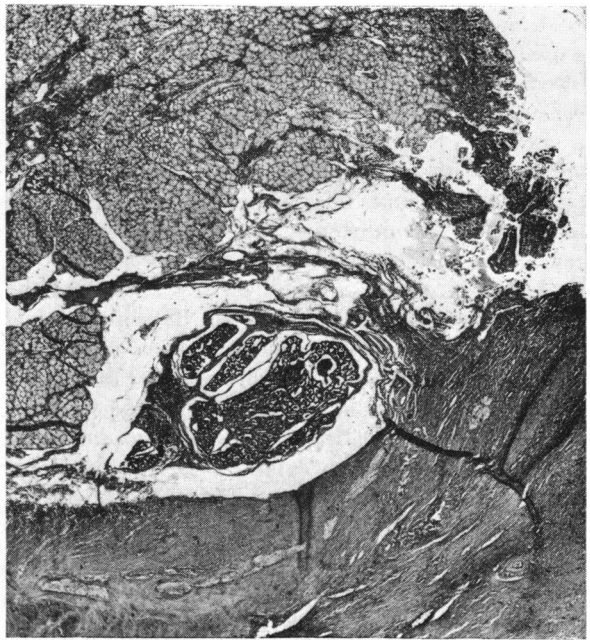

Figure 1.—Photomicrograph of the duodenal wall and the adenoma with the mucosa and the cluster of Brunner's glands above and the smooth muscle layers below. (Reduced from 16×.)

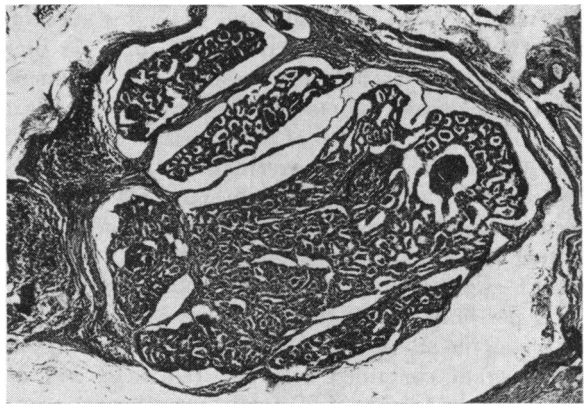

Figure 2.—Photomicrograph of the non-beta cell adenoma in the duodenal wall. (Reduced from 53×.)

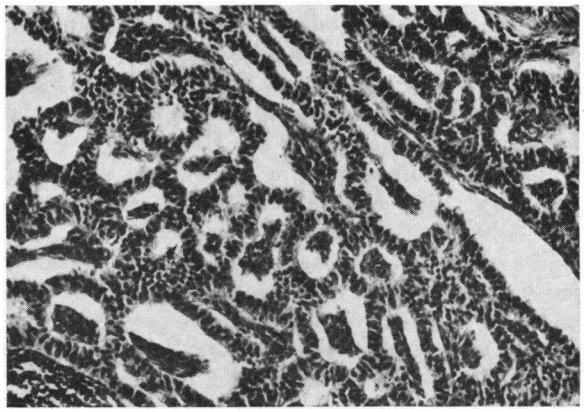

Figure 3.—Photomicrograph of the non-beta cell adenoma in duodenal wall. Lymphocytes in the left lower corner. (Reduced from 228×.)

acids and antispasmotics. A year after operation the patient had an episode of upper gastrointestinal bleeding requiring hospital admission and transfusions, and two years later, at the age of 42, she had a similar episode. Again at the age of 44 she was admitted with melena, weakness and fatigue. As the packed cell volume was 26 per cent four units of blood were administered. Roentgenologic studies revealed a gastric remnant which was considered to be rather large and a small ulcer just beyond the site of gastroduodenal anastomosis. Bleeding stopped spontaneously and the patient was discharged. Conservative management was continued and there were no further episodes of bleeding until 1963 when, at the age of 48, the patient again noted weakness and melena. She refused to enter the hospital and fortunately the bleeding stopped, but not until the hematocrit had dropped to 29. She did not receive a transfusion but the roentgenogram of the stomach after she had stopped bleeding again revealed a small ulcer of the duodenum just beyond the point of anastomosis. This ulcer was again noted after two months of diet and medication.

A gastric analysis revealed 16.6 mEq of free acid per liter and 15 mEq of total acid per liter in the fasting specimen; and following histamine stimulation (0.5 cc) the free acid rose to 47 mEq per liter with total acid of 60 mEq per liter. All specimens were free of bile. Three months later, in an effort to gain more knowledge of the secretory capacity of the stomach, a 12-hour, overnight gastric specimen which unfortunately contained some bile was collected. The total volume was 1,002 ml and it contained 13 mEq per liter of free acid (1.09 mEq per hour) and 30 mEq per liter of total acid (or 2.5 mEq per hour). This test was followed by a period of histamine stimulation (0.5 cc and although specimens again contained bile, the free acid was 31 mEq and the total acid 52.2 mEq per liter.

Atropine was then given and although all contained bile there was no free acid in any of three specimens, and the total acid was 14 mEq per liter. The serum potassium was 4.5 mEq per liter and the calcium was 4.5 mEq per liter. The fasting blood sugar was 93 mg per 100 ml, and the protein-bound iodine was 6.4 micrograms per 100 ml.

When last observed, the patient was following prescribed diet and medication and has had no further bleeding over a ten-month period.

The patients father had had ulcer symptoms for many years. He died as a result of an accident without having had any complication or operative treatment. Of her nine siblings, one brother had a duodenal ulcer which had bled a number of times. He had four operations for the ulcer and complications connected with the operations. He was, at last report, still having trouble. One sister had ulcer symptoms for many years and had massive bleeding on three occasions several years apart, care in hospital being required each time. She did not have surgical treatment.

Discussion

It is interesting that the existence of the adenoma in the wall of the first portion of the duodenum was not recognized by the operating surgeon nor was it described by the pathologist on his gross examination of the tissue removed. The exact size of this lesion was therefore never recorded, but it must have been very small. It was discovered in one of the microscopic sections of the duodenal wall, and a diagnosis of hamartoma of the duodenal wall, probably of pancreatic origin, was made. This was 18 months before the appearance of the original report by Zollinger and Ellison and seven years before Oberhelman's paper on ulcerogenic tumors of the duodenum, and the pathologist did not recognize it as an adenoma with a relation to the patients virulent peptic ulcer diathesis. Because further ulcerations developed after the operation, with four episodes of hemorrhage, a review of the microscopic sections was carried out, and the duodenal wall lesion was found to be consistent with an islet cell adenoma of the non-insulin secreting variety, considered by many investigators to be of alpha cell origin.[2]

One may speculate as to whether the patient had a functional ulcerogenic adenoma at the age of eight when she first started to have the upper abdominal symptoms that led to roentgenological diagnosis of an ulcer at the age of 13. It seems reasonable to assume that the ulcer diathesis in this patient, at least up to the time of operation, was based on the same physiological stimuli, and if that symptoms attributable to adenoma may have developed at an earlier age than in any other case thus far recorded. Davis and coworkers[1] reported the case of a boy, in whom symptoms started at the age of 15 years with a virulent form of ulcer diathesis, who eventually required total gastrectomy as the only satisfactory mode of therapy for a malignant non-beta cell neoplasm of the pancreas with metastatic tumor in the peripancreatic region.

The question does arise as to whether this lesion is malignant. There was nothing in the microscopic section to suggest malignant change in this case, and the subsequent course was not consistent with a malignant process. On the other hand, in three of the six cases reported by Oberhelman the lesions were malignant, as evidenced by local metastasis to lymph nodes. Oberhelman noted that the diagnosis of malignant change can not often be made by the histological appearance of the primary tumor; it is usually

made on the basis of metastasis to adjacent lymph nodes or to more distant sites. The incidence if malignancy in the ulcerogenic neoplasms in the pancreas itself is also approximately 50 per cent (Zollinger and coworkers[7]).

Jordan and coworkers[3] concluded from their own experience and that of others that the malignant lesions are not confined to the older age groups but are rather a disease of middle age with the greatest frequency in the fourth decade. The growth of the lesion, including metastatic implants, is slow and long survival is possible despite the presence of metastatic disease. Most but not all of the metastatic deposits function, and because of this the ulcer diathesis generally constitutes a greater risk to the patient than do the tumor and metastatic lesions. Total gastrectomy is indicated if there is evidence of unresected malignant deposits.

In the past ten years, the patient in the present case has had four major episodes of hemorrhage from a marginal ulcer in the duodenum which has been demonstrated by roentgenological studies. An understanding of the physiological mechanism behind her present tendency to form duodenal ulcers is of clinical importance. Is it a manifestation of additional adenomas either in the duodenal wall or in the pancreas, or of functioning metastatic lesions in the area, or does it represent the common form of marginal ulceration which relatively frequently follows subtotal gastrectomy of Billroth I type in patients with the usual form of peptic ulcer disease? These questions cannot be answered with any degree of certainty. It is known that ulcerogenic adenomas in the pancreas are often multiple, and presumably more than one can occur in the duodenal wall; indeed in one of Oberhelman's recent cases there were two duodenal adenomas.[4]

There are in the present case several reasons to doubt that the continued peptic ulcer activity was due to the presence of additional non-beta islet sell tumors of the duodenum or pancreas:

1. The ulcerogenic diathesis after the operation was perhaps not as severe as it might be expected to be if additional tumors were present.

2. Roentgenographic examinations reveal that there is a rather large gastric remnant still present. The recurrence rate in the case of the Billroth I procedure for duodenal ulcer without the addition of vagotomy is relatively high, especially if a high resection is not performed. Newton and Judd[6] in a recent follow-up study of 172 patients with gastric resection of Billroth I type found proved recurrent duodenal ulceration in 12.2 per cent of cases and suspected ulcerations in another 2.9 per cent; and recurrence was definitely more common in women. Thus small duodenal ulcerations that led to hemorrhage after the gastric operation in the present case may be simply owing to insufficient removal of the acid-secreting portion of the stomach. If this is so, adding vagotomy to the previous procedure might be sufficient to give her long-lasting protection from further trouble.

3. The studies on the gastric juice are suggestive that the continued trouble is not ascribable to retained ulcerogenic adenoma. Persons with an adenoma usually have a 12-hour nocturnal secretion of gastric juice exceeding 2 liters with a free acid content of 100 to 300 mEq, as compared with a normal volume in the range of 400 ml and an acid content of 18 mEq. A 12-hour nocturnal secretion of less than 100 mEq of hydrochloric acid may, however, be consistent with the diagnosis of an ulcerogenic tumor if the patient has had a previous gastric resection with or without a vagotomy, but many of these patients still produce large volumes of highly acid gastric juice. In the case reported here, the total volume was perhaps above average for a patient with a partial gastrectomy (1,002 ml) but the HCL output of 13 mEq during the 12-hour period is below what one might expect if an adenoma were still present. In addition, if an adenoma is present, often there is no definite response to histamine stimulation. (In the present case it was 10 mEq to 47 mEq per liter on one occasion; 13 mEq to 30 mEq per liter on another.) Also an inhibitory effect from atropine is not expected. (In the case here reported, no free acid was observed following atropine.) It would thus seem from all these observations that in the present case the patient's continuing difficulty may not be due to the presence of additional ulcerogenic adenomas but may represent the more ordinary form of marginal ulcer that can occur in any patient whose stomach continues to secrete moderate amounts of acid. The patient has been advised to have vagotomy but thus far has refused. If she is operated on at some time in the future, a very careful examination of the pancreas and the duodenum, preferably with the examining finger inserted into the lumen of the duodenum for more thorough bimanual palpation, should be performed. If no adenoma is found, vagotomy is probably the treatment of choice rather than the extensive procedure of total gastrectomy which is advised by many investigators when there is definite evidence of continued activity of one or more ulcerogenic tumors located in an area which can not be readily and completely removed.

Summary

A case is presented in which prolonged and severe ulcer diathesis was complicated by bleeding, obstruction and pain. Conservative treatment did not keep the disease in control. Incidental to partial gastrectomy with gastroduodenostomy an adenoma in the

wall of the first portion of the duodenum was removed. This lesion was morphologically consistent with the non-beta islet cell adenoma of the pancreas, the ulcerogenic tumor first described by Zollinger and Ellison. The patient had four episodes of hemorrhage from marginal ulcerations in the duodenum during a ten-year follow-up period after operation, but has had no further operation thus far.

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REFERENCES

1. Davis, C. E. Jr., Smith, P. Jr., and Davolas, X. S.: Ulcerogenic tumor of the pancreas, Ann. Surg., 155:669-680; 1962.

2. Greider, M. H., Elliott, D. W., and Zollinger, R. M.: An electron microscope study of islet cell adenoma, J.A.M.A., 186:120-123; 1963.

3. Jordan, G. L. Jr., Law, S. W., and Foster, R. P.: The management of patients with islet cell carcinoma exhibiting the Zollinger-Ellison syndrome, Amer. J. Surg., 105:80-86; 1963.

4. Oberhelman, H. A. Jr.: Personal communication to be published in Amer. J. Surg.

5. Oberhelman, H. A. Jr.; Nelson, T. S.; Johnson, A. N. Jr., and Dragstedt, L. R.: Ulcerogenic tumors of the duodenum, Ann. Surg., 153:214-227; 1961.

6. Newton, S. III, and Judd, E. S.: Long-term follow-up proves Billroth I gastric resection inadequate for permanent control of duodenal ulcer, S. G. & O., 116:170-174; 1963.

7. Zollinger, R. M., and Craig, T. V.: Ulcerogenic tumors of the pancreas, Am. J. Surg., 99:424-432; 1960.

8. Zollinger, R. M., and Ellison, E. H.: Primary peptic ulcerations of the jejunum associated with islet cell tumors of the pancreas, Ann. Surg., 142:709-723; 1955.

JUJUBE

A Cause of Perforated Bowel

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TO MOST PERSONS jujubes are a kind of hard gumdrops, with an unimpressive taste, that are bought for small money by small children. Some may have wondered casually about the origin of their musical name. In the present case, this curiosity was aroused and satisfied in a rather dramatic manner.

Report of a Case

The patient, a 57-year-old woman, a school teacher, was in excellent health except for mild hypertension, under good medical control, when the present episode started. She was teaching a class at school, when, at about 1 p.m., general abdominal cramping developed. As she knew that several of the other teachers had been bothered with diarrhea, she concluded that this was her trouble and in a few minutes excused herself, went to the bathroom and passed a soft stool. The cramps ceased for a time, but later in the afternoon recurred without any further stools, and by evening the cramping had turned into a rather colicky abdominal pain.

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The patient was examined and admitted to the hospital. A complete physical examination was in all respects uninformative. The abdomen was guarded during the attacks of colic but between the attacks was perfectly normal and painless to deep palpation.

Within the preceding year the patient had gastrointestinal and gallbladder x-ray examination that showed no abnormalities. The patient was observed in the hospital during the night and by morning the cramping attacks were more frequent and more severe and the patient was vomiting frequently. Results of urinalysis were within normal limits. Leukocytes numbered 20,000 per cu mm. X-ray films of the abdomen were taken and a consultant was called. The x-ray films showed laddering and fluid levels in the small bowel suggestive of obstruction. Physical examination was repeated and generalized abdominal guarding was noted, with definite rebound tenderness over most of the abdomen. The patient had no detectable hernia and there had been no previous abdominal operation. Fluids to make up the amount lost were administered intravenously and the abdomen was opened. The peritoneal cavity contained a considerable amount of thin, purulent material, with no odor, containing strands of fibrin and also particles of food. On further exploration a lesion was found at about the midpoint of the small bowel. Here a 5 cm segment of bowel was decidedly thickened and edematous, there was a tiny perforation at the mesenteric attachment. A hard foreign body could be felt within the lumen. It was approximately 3 cm in length and had sharp pointed ends. It was believed to be probably a swallowed bone. A 10 cm segment of bowel containing the foreign body, the inflamed area and the perfora-